extension or through hematogenous or lymphatic channels. Notwithstanding that great progress has been made in diagnostic methods, it still remains a fact that the most important part of our diagnostic information is derived from a careful study of a thorough history of the case.

In making a differential diagnosis of gall bladder disease, gastric and duodenal ulcers are among the conditions which call for the most careful consideration. Here, again, we find a carefully taken history of first importance, and in differentiating the intra-gastric diseases the most important aid which we have at our command is the x-ray. This gives us definite information in from 90 to 95 per cent of cases of gastric ulcer, provided it is in the hands of an expert roentgenologist. It is generally conceded, however, that the x-ray in gall-bladder disease furnishes confirmatory evidence rather than definite evidence. From our experience, I have found a careful study of the gastro-intestinal tract with a large series of x-ray plates is likewise of value in the diagnosis of gall-bladder disease, aside from the information furnished of intra-gastric conditions.

In our study of gall-bladder conditions, it is necessary to constantly keep in mind the fact, too, that the association of peptic ulcer and cholecystitis and chronic appendicitis is comparatively common and, in a certain number of cases, may be the cause of considerable confusion, especially where two or even three of these conditions are associated in the same patient.

STERLING BUNNELL, M. D. (Physicians Building, San Francisco)—The ideas expressed in this most commendable paper appeal to me as the true conception of cholecystitis. Instead of thinking in terms of gall-stone disease and recognizing only veteran cases, we are learning to recognize infections of the gall-bladder in their incipiency.

We now know that the infection is not limited to the gall-bladder, but is generalized in the biliary tract, through the gall-bladder ducts, pancreas, accompanying lymph glands and liver, and that it usually results from germ bombardment coming from infections in the portal system.

Removing the principal focus, the walls of the gall-bladder, by cholecystectomy, together with the original source of infection, if still present, usually cures, but for the following reasons it is not surprising that it does not always do so:

Several months to a year or more are necessary for the natural protective forces of the body to rid the rest of the biliary tract of infection, and especially so if deep-seated infection exists in the head of the pancreas or liver. Again, time is necessary for normal function to return in a stomach which has acquired the indigestion habit. Frequently enough after cholecystectomy the resulting adhesions pull on the pylorus or trigger-point of the stomach and cause a persistence of symptoms. Avoiding trauma in operation and routinely anchoring the end of the omentum in the duodenal-hepatic angle helps to prevent this.

Often tiny stones and sand in the common duct are overlooked, as it is impossible to palpate them through the duct walls. These cause a backfire of infection into the liver and continue the symptoms.

Disease of the gall-bladder makes its presence known to the clinician largely by a perversion of stomach functions, by general symptoms of infection, by disturbances of bile flow, by pain from duct obstruction, by pain, distress and tenderness from local infection in the gall-bladder.

The normal-acting stomach should handle the usual articles of diet and, if it does not, an organic lesion is often the cause. It is unsafe, though, to yield to our desire to simplify and assume that all dyspepsias are caused by the arch triumvirate, appendicitis, cholecystitis and peptic ulcer. When we consider what a multitude of diseases affect our outer covering, the skin, a tissue that we can see, we cannot but admit that our inner lining, the digestive tract, may also be subject to many different ailments.

However, when a patient tells of years of indigestion and of submission to prescribed diet lists and digestive aids, the real cause can usually be found in one or more of the above triumverate. If the indigestion is qualitative, intermittent, gradually increasing in severity and length of attacks and accompanied by symptoms of infection, both general and local, in the region of the gall-bladder, this organ comes decidedly under suspicion.

It is, however, only after a survey of all other symptoms, x-ray findings, physical signs and past history, and a due consideration of other possibilities, that we can arrive at a judgment of the case of sufficient value to advise surgical relief.

THE REFLEX NERVOUS DISORDERS AS DESCRIBED BY BABINSKI

By A. R. TIMME, M. D., *Los Angeles*

Sets up a group of nervous disorders which may not be classified as either functional or organic by usual methods. One case report.

These patients exhibit symptoms far more profound than could be accounted for on a functional basis, and yet not typical of any of the so-called peripheral or central lesions.

DISCUSSION *by Harold W. Wright, San Francisco; Samuel D. Ingham, Los Angeles; Edward W. Twitchell, San Francisco; Thomas J. Orbison, Los Angeles.*

WE HAVE long been accustomed to labeling as functional any case of paralysis that did not show classical symptoms of definite organic lesion of the nervous system. Thus, localization and grouping of signs indicated to us whether a lesion was in a peripheral nerve, in a plexus, spinal root, spinal cord, or anywhere up to and including the cortex, or in an artery, or in the meninges. Any case of paralysis or deformity that would not fit into any of the above molds was very likely to be classified as functional or hysterical.

There is, however, a group of cases, definitely organic, that do not fall into either of the above categories, i. e., these patients exhibit symptoms far more profound than could be accounted for on a functional basis, and yet not typical of any of the so-called peripheral or central lesions.

Although not explaining the pathology of the lesion, John Hunter, as early as 1835, called attention to muscular atrophy following joint affections, e. g., arthritis. Charcot and Vulpian in the eighties first predicated a reflex origin of such atrophies. Babinski, in the beginning of the Great War, finally gave us an adequate description of this type of lesion, in the form of papers, discussions, and an elaborate monograph. He terms the condition "nervous troubles of a reflex order" or "reflex nervous disorders" and postulates a reflex pathogenesis in certain cases of paralysis, contracture and deformity, atrophy, sensory and vasomotor change following wound or injury of an extremity that do not conform to any of the classical conceptions of central or peripheral lesion of the nervous system or to hysteria. Thus, an almost negligible wound of an extremity can give rise to profound changes in muscular tone and activity, can produce a disproportionate amount of atrophy with vasomotor and sensory disturbance, or it can produce a marked deformity or contracture. These changes cannot be explained by any of the known nerve, cord or brain lesions; they remain in a stationary or progressive state for years after the initial wound or injury has healed.

Prerequisite to the development of the above symptoms is the initial trauma or irritation, whether

an arthritis, a fracture of a long bone, or a gunshot wound. If a gunshot wound, it involves bone or soft tissues without injuring a nerve. Symptoms develop immediately after the injury or months later. They are out of all proportion to the severity of the injury, and in this respect resemble hysteria. They seem to disregard anatomical limits, but may be said roughly to appear in the same or adjacent spinal segments as the initial trauma. The following groups of symptoms are encountered:

(1) Muscular atrophy with paralysis. The atrophy is usually less marked than that of neuritis or anterior poliomyelitis and the paralysis only partial. It may involve single muscles, groups of muscles, or the entire extremity. The paralysis is of the flaccid type.

(2) Contractures and deformities. A few typical contracture deformities are described by Babinski, e. g., contracture of the pelvi-trochanteric muscles following traumatism in the region of the hip, producing a typical "salutation gait"; flexion contracture of the leg, in which the heel remains raised both in standing and walking; various types of club-foot and claw-toes; numerous forms of contractures of the hand and fingers, including the typical "accoucheur's hand"; hypertonus of the flexors of the hand associated with hypotonus of the extensors, producing a strong wrist-drop deformity; and many others.

(3) Vasomotor and temperature changes. The affected limb is blue and cold and responds to air or water temperature variations more slowly than the sound limb. It may also be mottled and reddish and pit on pressure. Arterial pulsations may be weaker in the affected member and the oscillations of decreased amplitude. Obstinate coldness of the hand or foot is the rule in cold weather.

(4) Hyperexcitability of the muscle to percussion, together with slowness of the muscular contraction resulting, are described by Babinski. On striking the belly of the affected muscle with a small percussion hammer, a contraction of great amplitude is produced, which is sustained longer than normal. Babinski has demonstrated this graphically.

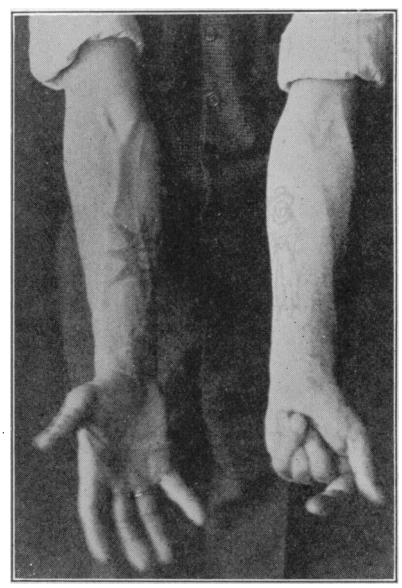
(5) Loss of muscular tone may be pronounced in the affected muscle groups. Thus the thigh may be hyperflexed on the body, with the leg extended, or with the leg itself hyperflexed on the thigh, producing an exaggerated letter "Z."

(6) Electrical changes. There may be increased response, i. e., contractions of increased amplitude to both galvanic and faradic currents, or there may be a corresponding hypoexcitability. Babinski also describes premature fusion of contractions to the faradic current. True R. D. is never present.

(7) Changes in reflexes. Tendon reflexes are normal or exaggerated on the affected side. Sometimes the exaggeration can be demonstrated only while the patient is anesthetized.

(8) Sensory changes are inconstant and may assume the form of a hyper or hypoesthesia, frequently of a segmentary distribution. Spontaneous pain in the affected area is also frequently encountered.

In differential diagnosis the chief source of trouble is hysteria, since these cases do not conform to the symptomatology of any of the known anatomical lesions. A valuable aid in establishing a diagnosis of reflex disorder is examination during chloroform anesthesia. In this state the tendon reflexes become definitely exaggerated on the affected side, attempt to correct the deformity causes muscular spasm, and the contracture persists to an advanced stage of the narcosis. These signs are not true of hysteria. The deformity of reflex disorders also persists during sleep. Other differences are as follows: The paralysis in reflex disorders is more limited in extent and more persistent; the deformities do not correspond to any natural attitude as they do in hysteria; atrophy, vasomotor, and trophic changes are more



marked in reflex disorders; changes in muscular tone are more pronounced than in hysteria; electrical reactions and reflexes are normal in hysteria, but affected in reflex disorders.

Differentiation from organic monoplegia and from peripheral neuritis is easier, since it calls into play our knowledge of anatomy and nerve distribution. The well-known Babinski reflex is never present in these cases, nor are any of the typical signs of upper-motor-neurone lesion. The location of pain, wasting of muscles, R. D. and correlation of motor and sensory symptoms serve to distinguish a peripheral neuritis.

Volkmann's ischemic paralysis and Dupuytren's contracture sometimes produce deformities similar to those of reflex disorders, but the muscular induration and lack of electrical reaction in the Volkmann lesion, the nodular change in the palmar skin and fascia in the Dupuytren lesion serve as valuable diagnostic points.

As to pathogenesis, Charcot first postulated a reflex origin of the atrophy incident to an arthritis—a peripheral irritation of centripetal nerves in the joint by the inflammatory process, causing an irritation of the anterior horn motor cells, with a resultant atrophy. This reflex arc is called into play in the cases under discussion, Babinski maintains, and so not only furnishes a rational explanation of causation, but also includes these disorders in the organic category. As yet, however, actual histologi-

cal change in the anterior horn cells has not been demonstrated.

PROGNOSIS AND TREATMENT

Since reflex disorders are an undoubted organic condition, psychotherapy has no place in their treatment, except insofar as hysterical symptoms are superimposed. Babinski reports improvement following gentle massage and movement and continuous extension. He cautions against violent and energetic means which are likely to increase the deformity by causing further irritation of the reflex arc. He reports favorable results with mild applications of heat and diathermy. Certain heroic measures have been tried, such as alcohol injections into nerves, excision of the scar tissue of the initial trauma, a destruction of the periarterial plexuses of sympathetic filaments, all with more or less indifferent results.

CASE REPORT

W. R. W. Age 40. Received a through and through wound of the left wrist by a machine gun bullet in December, 1915. The bullet entered the dorsum of the wrist near the head of the radius and emerged from the palmar aspect near the base of the last metacarpal. No infection occurred and the scars are at present barely visible. A plaster cast was applied the following day and remained on about three months without causing any undue discomfort. The cast extended from the base of the metatarsals half-way to the elbow. Flexion contracture of the fingers began almost immediately following the injury, and when the cast was removed the fingers were half closed. This contracture has been progressive to date, in spite of the application during 1917 and 1918 of a large palmar splint designed to maintain the fingers in extension. The flexor pull of the fingers against this splint caused marked pain.

Today the last three fingers of the left hand are so tightly drawn against the palm that the distal ends of the middle phalanges bid fair to crowd through the skin of the fingers. There is beginning slight erosion of the skin of the palm. The third and fifth fingers override the fourth. Forcible extension of the fingers away from the palm is impossible to an extent greater than one inch. Motion at the wrist, of the thumb and index finger is practically unimpaired. There is slight atrophy of the hypothenar eminence. There is one-quarter inch atrophy at the wrist and one-half inch at the forearm. Other movements of the upper extremity are unimpaired. The tendon reflexes are equal to those in the right arm. There is no R. D., in fact, response to the faradic and to K. C. C. is even more prompt than in the right forearm. Direct muscular excitability is about equal in the two forearms and in the two thenar eminences. There is no sensory change. There is obstinate cyanosis and hypothermia in cold weather and after immersion in cold water. There is, at such times, decreased amplitude of the radial pulsations.

General physical and neurological examination is otherwise negative.

Other conditions to be considered in this case are: (1) ulnar neuritis, since the ulnar side is chiefly involved, (2) Volkmann's ischemic paralysis, (3) Dupuytren's contracture, (4) hysteria. Ulnar neuritis is ruled out by absence of typical atrophy and sensory change. Volkmann's is ruled out by absence of muscular induration and presence of electrical reaction. There is no evidence of hypertrophy of the palmar fascia or ridging of the skin of the palm, such as is seen in Dupuytren's disease. As to hysteria, it is inconceivable that a deformity of this severity or duration could be entirely functional. Furthermore, the positive vasomotor changes, the gradual progression of the deformity during eight years, the unnaturalness of the deformity, i. e., the impossibility to assume the position voluntarily, all point to an organic affection.

Brockman Building.

DISCUSSION

HAROLD W. WRIGHT, M. D. (Flood Building, San Francisco)—This paper is an important contribution as a clear summary of these forms of peculiar syndromes, following peripheral injury and because it calls attention forcibly to the features which distinguish such disorders from hysteria. The word "reflex," as applied to such symptoms and pathological conditions, is, to my mind, not very satisfactory. Nervous reflexes of manifold and peculiar nature, producing symptoms of disordered function at a distance from the place of origin of the reflex activity, we are familiar with, but such reflex disorders are disorders of function and not of structure, i. e., not lesions. In the conditions described in this paper we have definite pathological changes of tissue resulting from something besides a mere irritation of function. May it not be that in some of these disorders there has been an unrecognized infection of the end plates of motor nerve filaments, a localized neuritis at first, then irritative reflexes resulting, and possibly an ascent of the infective or toxic process to anterior horn cells with resulting irritation of other reflex paths at that level?

SAMUEL D. INGHAM, M. D. (1920 Wilshire Boulevard, Los Angeles)—The diagnosis of the conditions here presented offers much difficulty, since it is well established that certain of the functional cases, with local paralysis and contractures develop tissue changes, including joint fixations and rarefication of bone, as secondary results. On the other hand, indefinable organic and toxic conditions may affect the physical integrity of the nervous structures without producing clean-cut signs or symptoms. We frequently see atrophy of muscles that have been subjected to prolonged reflex rigidity, and that is apparently the conception of Babinski in regard to these cases.

It is impossible to say, in view of our present knowledge, whether there may not be, as Doctor Wright suggests, an absorption of toxin or even actual infection of the peripheral nerves with sufficient resulting disturbance of the innervation to explain the symptoms.

EDWARD W. TWITCHELL, M. D. (909 Hyde Street, San Francisco)—The word "reflex" is very unsatisfactory to describe the condition, and it is curious that Babinski, with his genius for naming symptoms, did not devise a name free from the old associations inseparable from "reflex." He was alive to the possibility of the role that the autonomic system might play in these cases and spoke of it in his monograph. That seems to me to be the most nearly adequate explanation for the present. If a causalgia be the expression of the sensory manifestations of sympathetic diseases, why may not this irritation extend to the trophic and motor fibers of the same system? Why is a contracture in the case described not due to stimulation of a reflex arc contained entirely within the system of sympathetic fibers efferent and afferent?

The paper is important in that it jars us out of our dimensional attitude toward nervous diseases and brings to our notice another dimension to which others will, no doubt, in time be added.

THOMAS J. ORBISON, M. D. (2007 Wilshire Boulevard, Los Angeles)—I do not disagree with the diagnosis made by Timme in this very interesting and well-presented case. On the contrary, I am inclined to concur in it. But there are two or three points that need to be cleared up. One is that this man has never been subjected to chloroform anaesthesia—at least no mention has been made of this. It would seem to me that this would give the real clue that would enable a correct diagnosis.

The other point is that I noticed that quite a marked extension could be obtained when, in my examination of the patient, moderate, but sustained traction in extension was made on the fingers. This seemed to tire, and I noted a more or less rhythmical attempt at maintaining the original posture, which attempts came with less force at the end of the short time I made traction. A third point should be borne in mind, namely, that his hand remained in a splint for three months. In my opinion

this man should have been anesthetized quite early in order to make diagnosis certain.

DOCTOR TIMME (closing)—The strongest criticism encountered by Babinski, when he formulated this concept, was from those who clung to the hysterical explanation for such cases as these. But in this case hysteria would be a diagnosis by elimination, rather than a positive one. We have all been too prone to call a thing functional if it does not fit into the known organic pictures. After all, the tendency of medical research is to increase the number of "organic pictures."

Among many wounds of extremities in American troops, I have encountered only two of these cases, whereas the French found them with much greater frequency.

I have been unable to find any pathological reports whatever, but I have no doubt that some alteration in structure will be seen somewhere between anterior horn cells and motor end-plates.

The sympathetic fibers are, no doubt, always involved, as shown by the vasomotor and trophic changes. But there is no evidence as yet that striped muscle has any sympathetic innervation, or at least that sympathetic stimuli can produce striped muscle spasm.

This man had moderate and sustained traction by means of splints for nearly two years, but the deformity persisted and advanced. This speaks for a powerful irritative factor at work. A splint deformity does not act this way.

Let us keep this concept of reflex disorder in mind. Perhaps many doubtful and borderline cases can, in the future, be so explained.

A Clinical Classification of Bright's Disease—Thomas Addis, San Francisco (Journal A. M. A.), made a study of the effect of various physical factors on the formed elements of the urine. He found that hyaline casts and all casts whose matrix was hyaline disappeared from neutral sodium chlorid solutions when the salt concentration was reduced to less than 0.5 per cent. When the hydrogen ion concentration of the solution was varied, it was observed that the less the hydrogen ion concentration the greater was the concentration of sodium chlorid necessary to keep the casts from dissolving. He had already noticed that those specimens of urine in which few or no casts were present were either dilute or alkaline or had both of these properties in lesser degree. It seemed likely, therefore, that variability was no inherent characteristic of cast formation, but might be the result of changes in the degree of dilution and reaction of the urine. This proved to be the case, since the variability was replaced by a satisfactory degree of constancy whenever the conditions were such as to induce the secretion of concentrated and acid urine. The results of the examination of the urinary sediments of the patients who were investigated fall, naturally, on qualitative as well as on quantitative grounds, into three main divisions; and since these divisions seem to be corroborated by many significant facts elicited by other methods of examination, Addis has become persuaded that they represent three diseases that are pathologically and etiologically distinct and separate. In order to avoid the confusion that would result from the use of terms that are familiar but have been used with a somewhat different meaning, he has reverted to a purely descriptive terminology and has called the first division hemorrhagic Bright's disease (seventy-one cases), the second degenerative Bright's disease (forty-three cases), and the third arteriosclerotic Bright's disease (twenty-six cases). The initial stage is a sequel of streptococcal infections. The second disease has been called degenerative Bright's disease because the constant and most prominent feature of the sediment is the large number of epithelial cells in various stages of granular or fatty degeneration. The third disease, arteriosclerotic Bright's disease, is of great importance because it occurs more frequently than any other form of Bright's diseas. But so far as the kidney is concerned, its importance is of a negative character; for when the diagnosis has been made it is no longer necessary to consider the renal lesion as a factor in the management of the patient. These are the patients with hypertension who are often told that they are suffering from "chronic interstitial nephritis" and who consequently live in fear of death in uremia that never comes. These conditions are described in detail.

Clinical Notes and Case Reports

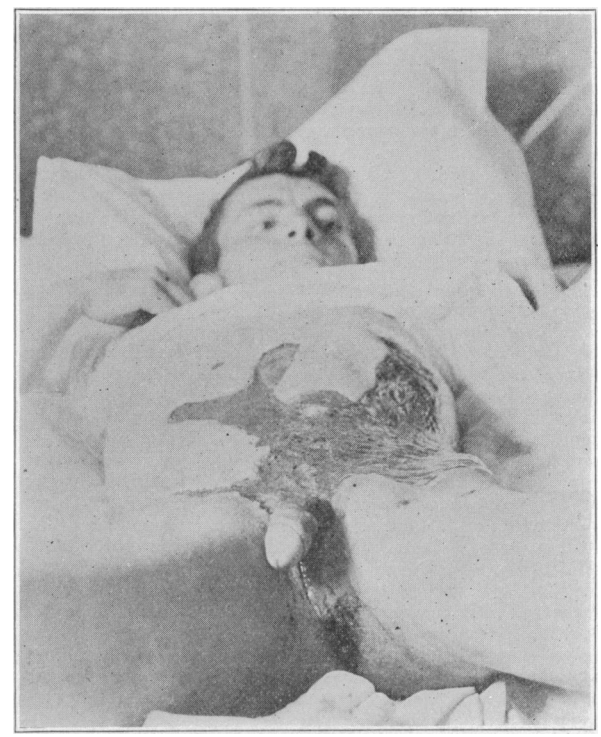
UNUSUALLY EXTENSIVE INJURY

REPORT OF A CASE

By S. M. SPROAT, M. D., *Portola, California*

A. O., age 22. Mill laborer, single, was brought to the Sierra Valley Hospital at Loyalton July 28, 1925, suffering from avulsion of the entire scrotum, skin, and subcutaneous tissues of the entire penis, abdomen, and left side, with testicles entirely denuded, and lying on the abdomen. The patient had been caught in the live rollers of the mill at 7:45 a. m., and was bleeding profusely and suffering profoundly from shock when seen at 10 a. m. in consultation with Dr. W. A. Lavery of Loyalton. Pulse 135, weak, thready; blood pressure, systolic, 90.

The patient was at once removed to the operating-room, anesthetized, and the entire area scrubbed with a scrubbing-brush and tincture green soap and water, after which sterile water was poured over the entire area. The testicles were placed in an artificial scrotum formed



by flaps from the inner sides of the thighs. The flaps were united in the midline with sutures of silkworm gut, leaving the perineum resembling that of a female. The penis was repaired and covered with skin drawn back from the foreskin which covered the distal end of the penis and which had escaped injury. The proximal end of the penis was covered with skin which had been avulsed from the abdomen and placed around it in a spiral manner. This graft did not live. The abdomen was covered, to a large extent, by bringing in large skin flaps from the surrounding healthy skin, and also by one large flap, which had been torn off. The skin which had been avulsed did not live, nor was it expected that it would live at the time of operation, but it was thought advisable to cover as much raw area at this time as possible on account of the likelihood of this lessening, to some extent, the marked shock which was present, and it evidently served this purpose.

For about ten days the patient had a stormy conva-